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Date: Friday, March 4, 2016

Time: 12:45–14:15

Room: Hall 3 (Posters &amp; Exhibition)

**HIV missed . . . CMV shows the way**K.A.C.G. Gandhi<sup>1,\*</sup>, S. Mankar<sup>2</sup>, B. Purandare<sup>2</sup>, S. Ialwani<sup>2</sup>, J. Oswal<sup>3</sup>, V. Kalrao<sup>3</sup><sup>1</sup> Bharati Hospital & Research Centre, PUNE, Maharashtra, India<sup>2</sup> Bharati hospital, Pune, India<sup>3</sup> Bharati hospital, pune, India

**Background:** We present a case of transplacentally acquired human immunodeficiency virus (HIV) infection in a child who presented with disseminated cytomegalovirus (CMV) infection

**Methods & Materials:** We present a case of transplacentally acquired human immunodeficiency virus (HIV) infection in a child who presented with disseminated cytomegalovirus (CMV) infection. A 16 months old female child presented with complaints of failure to thrive, regression of developmental milestones and 2 episodes of convulsions since 10 months of age. Child was well till age of 10 month followed which started showing regression of milestones like walking with support, standing and sitting without support, speaking bisyllables which she was able to do before. Child had signs of severe acute protein energy malnutrition like dry thin brittle hair, dry skin. Examination also revealed microcephaly, icterus, oral thrush, no eruption of primary teeth and hepatomegaly.

**Results:** Complete blood count showed severe anaemia (4.7gm) and thrombocytopenia (78000) and leucocytosis (18400). Renal function tests (Sr. creatinine=2.1, blood urea=71) and liver function tests (SGOT=1231, SGPT=258 serum bilirubin=4.8) were found to be deranged. Ophthalmoscopic examination was strongly suggestive of Cytomegalovirus retinitis. Infection was confirmed by a positive result for CMV on polymerase-chain-reaction analysis of blood and urine. With the clinical picture of disseminated CMV infection, immunodeficiency was strongly suspected. HIV DNA PCR of both child and mother was positive for HIV-1. Absolute CD4 count of child was 298 and percentage was 33%. To combat active retinitis, intravenous ganciclovir was started and planned to start antiretroviral treatment 2–3 weeks later.

**Conclusion:** Early detection and prompt treatment of HIV and associated opportunistic infections is of utmost importance. Paediatric HIV/AIDS can present as failure to thrive and regression of acquired milestones. Initiation of highly active antiretroviral therapy should be done after adequate control of underlying opportunistic infections to prevent immune reconstitution inflammatory syndrome.

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**HIV-1 pol gene polymorphism and transmitted drug resistance (TDR) in chronically infected HIV-1 antiretroviral treatment naïve patients in South India**S. Gomathi<sup>1,\*</sup>, S. Sivamalar<sup>2</sup>, T.R. Dinesha<sup>3</sup>, J. Boobalan<sup>4</sup>, P. Balakrishnan<sup>5</sup>, A. Pradeep<sup>5</sup>, S. Poongulali<sup>5</sup>, S.S. Solomon<sup>5</sup>, S. Solomon<sup>5</sup>, S. Saravanan<sup>5</sup><sup>1</sup> YRG CARE, Chennai, India<sup>2</sup> YRG CARE infectious Diseases Laboratory, Chennai, Tamil Nadu, India<sup>3</sup> YRG CARE, Chennai, Tamil Nadu, India<sup>4</sup> Y.R. Gaintoind Centre for AIDS research and Education, Chennai, Tamilnadu, India<sup>5</sup> YRG CARE, Chennai, India

**Background:** India with 2.1 million HIV populations, good access to first-line antiretroviral therapy is widely available, however understanding HIV Transmitted Drug Resistance (TDR) and polymorphism is critical for continued success.

**Methods & Materials:** HIV-1 pol gene spanning 20–240 codons of RT was genotyped by validated homebrew method for 100 ART naïve participants. The sequences were analyzed according to WHO recommendations using the Calibrated Population Resistance (CPR) tool of Stanford University HIV drug resistance (DR) database. For polymorphism identification subtype C consensus was used. Sequence was aligned (Clustal X) to an Indian subtype C reference C.IN.AF067155) and examined for HIV-1 subtype using REGA V3. Majority were subtype C infected 99% (n=99).

**Results:** Among 100, 61 were male, with median age and CD4 count of 37 years (IQR 27–38), and 244 cells/uL (153–359) respectively. TDR was observed among 4 participants, which is considered to be low level based on the WHO recommendations for public health action in response to TDR survey results. Observed mutations were M41L (1%), K219R (1%), K101E (1%) and Y181C (1%). The observed NRTI DRMs has less effect on available NRTI options while NNRTI DRMs will have adverse impression on available NNRTI options. Apart from TDR, naturally occurring polymorphism is also a main concern as some polymorphism may cause resistance to ART (eg. V90I, V118I, E138A, V179D). Based on that all the patients had polymorphic mutations and the predominant polymorphisms observed were A36E (41%), K211R (36%), A162S (35%), in addition polymorphic mutation causing resistance such as V90I, V118E, E138A and V179D was seen in 2, 1 and 3 individuals respectively. If subtype B consensus was taken for polymorphism identification, positions of RT such as 35, 36, 39, 48, 60, 121, 122, 162, 173, 177, 200, 207 and 211 would be overestimated as >60% polymorphism in that position.

**Conclusion:** We conclude, low prevalence of HIV DR among naïve participants. In addition 17 positions known to be polymorphic in subtype B (between 20–240 codons) has different amino acid consensus in subtype C. Thus polymorphisms pertaining to subtype C should be functionally annotated for DR.

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